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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

APPLICANT: Frederickson et al	§	**ART UNIT:**
	§	**1617**
FILED: March 22, 2004	§	
	§	**EXAMINER:**
SERIAL NO.: 10/805,882	§	**Huynh Carlic K**
	§	
FOR: Reduction of Zinc-induced Neurotoxic Injury by Blockade of Nitric Oxide Synthesis	§	
	§	
	§	**DOCKET:**
	§	**D6489**

MS NON-FEE AMENDMENT
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

CERTIFICATE OF MAILING UNDER 37 CFR 1.8

Dear Sir:

I hereby certify under 37 CFR 1.8 that the following correspondence is being deposited with the United States Postal Service as first class mail with sufficient postage on the date indicated below and is addressed to Mail Stop AF, Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450.

1) Response to Restriction Requirement

Please return the enclosed postcard acknowledging receipt of this correspondence.

Respectfully submitted,

Date: May 29, 2007

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Counsel for Applicant

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RESPONSE TO RESTRICTION REQUIREMENT

Dear Sir:

In response to the Restriction Requirement in the Examiner's communication mailed April 30, 2007, Applicants hereby elect, without traverse, Group I, claims 1-6, drawn to a method of inhibiting zinc release from neurons. In response to the species election, the Applicants, provisionally elect species 1, directed to several different agents that inhibit nitric oxide synthesis (including iNOS and nNOS) with traverse. Further, the Appliants request that species 2, directed to an agent that increases eNOS activity be joined with species 1 for examination. The Examiner contends that these are patentably distinct species. Applicants disagree.

Applicants submit that the instant invention is directed to controlling neurotoxic zinc release by inhibiting synthesis of nitric oxide. The enzyme that catalyzes the formation of NO from oxygen and arginine is nitric oxide synthase. There are three isoforms of nitric oxide synthase, i.e., iNOS, nNOS, and eNOS.

Zinc release from the neurons can be modulated through the regulation of nitric oxide synthesis via all three nNOS, iNOS and eNOS enzymes. The instant invention inhibits NO production by inhibition of nNOS and iNOS leading to inhibition of zinc release. At the same time to augment cerebral blood flow eNOS is activated or alternatively a pressor (e.g., the a- and b-adrenergic catecholamine dopamine) can be used. The instant invention demonstrates that a combination of decreased zinc, due to inhibition of iNOS and nNOS, and enhanced cerebral blood flow, due to activation of eNOS or by using a pressor, leads to effective prevention of zinc-mediated brain injury. Hence, the effectiveness of the methods of the instant invention lies, at least in part, in a combined use of species 1 and 2. Although, structurally species 1 and 2 encompass distinct compounds, their intended use, as disclosed by the instant invention is the same, i.e., regulation of nitric oxide synthesis. Accordingly, the Applicant respectfully requests that for examination species I, drawn to several different agents that inhibit nitric oxide synthesis be joined with species 2, drawn to several different agents that increase eNOS activity.

Respectfully submitted,

Date: May 25, 2007

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